Medical Management of Aortic Valve Disease

JOEL KARLINER, MD*

IN GENERAL, all patients with aortic valve disease should be counseled regarding the necessity of paying particular attention to the state of their general health, and should be apprised of the symptoms of endocarditis and the necessity for, and details of, endocarditis prophylaxis. In patients with aortic stenosis who are without the typical symptoms of angina pectoris, syncope or congestive cardiac failure, intelligent observation and *careful* follow-up are generally all that are required. Patients must be cautioned to report any symptoms to the physician, and yearly or more frequent x-ray studies of the chest, electrocardiograms and determinations of systolic time intervals are indicated to assess the progression of aortic stenosis and left ventricular hypertrophy. Asymptomatic patients with suspected significant aortic stenosis should be cautioned to avoid exercise which is strenuous or excessive, including jogging and competitive sports. Such limitation of vigorous activity need not apply to a person with mild to moderate disease. Although treadmill or bicycle exercise testing may be helpful in defining symptoms more accurately in individual patients, particularly those who are asymptomatic but with suspected significant stenosis, such procedures are potentially dangerous and should be carried out with caution. If doubt persists as to the severity of the lesion, cardiac catheterization is indicated.

Whether or not digitalis glycosides should be prescribed for patients with left ventricular hypertrophy of any cause is uncertain. As discussed by Drs. Peterson and Tsuji, while some evidence indicates that the process of hypertrophy in aortic stenosis may be associated with depression of left ventricular performance, recent experimental work indicates that this is probably not the case, at least during the early stages of left ventricular hypertrophy.[12] There is no evidence in humans that digitalis therapy in this setting can delay the progression of left ventricular hypertrophy. In patients with significant aortic stenosis it is uncommon for electrocardiographic findings to be normal[4,40] and administration of digitalis may induce repolarization changes in electrocardiograms which might complicate the diagnosis of left ventricular subendocardial ischemia and hypertrophy. Therefore, on balance, digitalis glycosides probably should not be used prophylactically in patients with aortic stenosis and left ventricular hypertrophy unless congestive cardiac failure is present, or reduced left ventricular performance can be demonstrated by noninvasive methods.

Diuretics should be employed in the treatment of congestive cardiac failure, but with appropriate caution, since vigorous diuresis may lead to orthostatic hypotension and fatal arrhythmias.[29] Such an occurrence may be expected in patients with aortic stenosis because in such patients the use of the Frank-Starling mechanism is required and often an elevated left ventricular end-diastolic pressure to achieve an adequate cardiac output is needed. When end-diastolic volume is reduced beyond a critical level by diuretic medication, the cardiac output may decline, resulting in inadequate coronary or cerebral perfusion, or both. In this situation, however, strong consideration should be given to cardiac catheterization and aortic valve replacement.

Other conventionally employed drugs should also be used with caution. For example, propranolol can depress myocardial function and induce left ventricular failure; it may reduce the heart rate response to exercise, leading to inadequate cardiac output and to decreased perfusion of vital organs. Furthermore, bradycardia is poorly tolerated in patients with aortic stenosis, as it causes the mean systolic gradient across the aortic valve to increase. Similarly, any antihypertensive agent (such as guanethidine) which depresses myocardial performance should not be used in patients with aortic stenosis.

Patients with chronic aortic regurgitation typically tolerate this lesion well and tend to be free of symptoms until relatively late in the course of disease, usually *after* the development of myocardial dysfunction.[5] Therefore, the goals of a physician in managing patients with aortic regurgitation are to: (1) prevent or postpone, if possible, the myocardial dysfunction that so insidiously complicates the course of chronic aortic regurgitation and (2) detect the earliest signs of myocardial dysfunction in an effort to properly time aortic valve replacement. Pertinent to these considerations, several important clinical questions remain unanswered. A question regarding the first of these goals is whether use of digitalis preparations early in the course of chronic, significant aortic regurgitation will decrease the amount of

*Associate Professor of Medicine.

left ventricular dilatation, and thereby prevent or postpone the onset of myocardial disease. Our bias is toward use of digitalis preparations in a patient with left ventricular dilatation, as assessed by clinical, radiographic or echocardiographic techniques, before the onset of either overt depression of systolic ventricular performance or of significant symptoms of heart failure. Certainly, once symptoms of circulatory congestion appear, aggressive therapy with digitalis glycosides, diuretics and sodium restriction is indicated. Again, considering the goal of preventing myocardial dysfunction, systemic arterial hypertension is a particular problem in a patient with aortic regurgitation, because it increases regurgitant volume and systolic wall stress, and the natural history of aortic regurgitation might thereby be accelerated. Therefore, as discussed by Dr. Engler, significant hypertension should be appropriately treated. However, the use of drugs that might decrease the inotropic state of the ventricle or result in bradycardia—such as propranolol, reserpine or guanethedine—should be avoided.

Atrial fibrillation, while uncommon in isolated aortic regurgitation, may be poorly tolerated if atrial systole is an important contributor to end-diastolic volume in a given patient, and cardioversion may be required. Bradyarrhythmias may also be detrimental and may require the use of a permanent pacemaker to reverse the increase in left ventricular volume and pressure caused by long diastolic periods.[4]

Exercise, in general, is well tolerated, as it is accompanied by an increase in heart rate, and a decrease in aortic impedance, both of which reduce the volume of aortic regurgitation.[42] However, any situation which provokes symptoms should be closely evaluated and avoided if it appears to be causing an excessive hemodynamic burden.

Indications for and Objectives of Cardiac Catheterization in Aortic Valve Disease

ALLEN D. JOHNSON, MD

THE TIMING of cardiac catheterization in aortic stenosis, in general, is related to our knowledge of the natural history of this disorder. Once angina, congestive heart failure or otherwise unexplained syncope occurs in a patient with significant aortic stenosis, life expectancy is significantly abbreviated in the absence of surgical relief of the valvular obstruction.[4,21] When any one of these symptoms is present, cardiac catheterization, in anticipation of aortic valve replacement, is indicated. When doubt exists regarding the severity and relative contribution of aortic valve disease in a patient with angina, suspected coronary artery disease, calcium in the region of the aortic valve and a systolic ejection murmur (not an uncommon clinical situation), cardiac catheterization is often necessary to establish the presence and severity of aortic stenosis, and to plan appropriate surgical or medical therapy. More controversial is the decision regarding cardiac catheterization in an asymptomatic patient with suspected severe aortic stenosis. Approximately 5 percent of such patients beyond adolescence will die suddenly without symptoms.[21] However, since operative mortality is in the same range, we prefer to postpone catheterization and surgical therapy until symptoms are present.

The goals of cardiac catheterization in aortic stenosis are:

- *To document the severity of the aortic obstruction.* This requires accurate measurement of the cardiac output simultaneously with measurement of the left ventricular and systemic arterial (preferably ascending aortic) pressures. The aortic valve can usually be crossed retrogradely from the brachial or femoral approach; however, transseptal catheterization of the left ventricle is occasionally required and is particularly useful in such a case. Using the Gorlin formula (aortic valve area = systolic flow/K $\times$ $\sqrt{\text{gradient}}$), the approximate aortic valve area can then be calculated, assuming there is insignificant aortic regurgitation. Careful attention should be directed to the interpretation of the peak and mean systolic aortic valve pressure gradients, as the gradient will vary directly with the square of the systolic blood flow across the valve per unit time, and a low cardiac output could be associated with a low